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Chronic-Phase Chronic Myeloid Leukemia in a 30-Year-Old Woman Presenting with Aquagenic Pruritus and Persistent Leukocytosis: A Case Report

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ABSTRACT: Chronic myeloid leukemia (CML) is uncommon in young adults and may initially resemble reactive leukocytosis. We describe a 30-year-old woman evaluated for persistent leukocytosis and thrombocytosis accompanied by aquagenic pruritus, drenching sweats, and severe fatigue. An empiric antibiotic trial produced little change in her leukocyte count. Peripheral blood examination showed a granulocytic left shift with basophilia and few circulating blasts. Bone marrow examination showed approximately 95% cellularity, granulocytic predominance, atypical megakaryopoiesis, and no increase in blasts. Molecular testing detected p210 BCR::ABL1 e14a2 and e13a2 transcripts, establishing chronic-phase CML. Imatinib 400 mg daily produced hematologic improvement and a substantial decline in BCR::ABL1 transcript levels. However, musculoskeletal pain followed by persistent diarrhea, occasionally accompanied by fecal incontinence, interfered with daily activities and prompted a switch to asciminib 40 mg twice daily. At the October 2026 follow-up, gastrointestinal symptoms had resolved and early tolerance of asciminib was favorable. This case highlights the importance of evaluating persistent myeloid-pattern leukocytosis regardless of patient age and balancing molecular response with treatment tolerability during long-term CML care.

KEYWORDS: chronic myeloid leukemia, young adult, aquagenic pruritus, BCR::ABL1, tyrosine kinase inhibitor

Introduction

Chronic myeloid leukemia (CML) is a clonal myeloproliferative neoplasm driven by the BCR::ABL1 fusion gene, most commonly resulting from the Philadelphia chromosome translocation t(9;22) [1]. Although CML can occur at any age, it is predominantly diagnosed in older adults. According to the United States Surveillance, Epidemiology, and End Results (SEER) program, the median age at diagnosis is 67 years, and only 6.7% of new cases occur among individuals aged 20-34 years [2]. Studies of younger patients show that CML is an established, although less frequent, diagnosis in this age group [3]. A young patient's age should therefore not outweigh persistent findings suggestive of a myeloproliferative disorder.

CML may be discovered incidentally through routine blood testing or present with fatigue, night sweats, weight loss, or abdominal discomfort [1]. The diagnostic evaluation of leukocytosis must consider whether the elevation is reactive or clonal. Persistent neutrophilic leukocytosis accompanied by a granulocytic left shift, basophilia, or thrombocytosis warrants peripheral smear review and, when appropriate, hematology evaluation [4]. Confirmation of CML requires detection of BCR::ABL1, while marrow morphology and cytogenetic assessment help characterize the disease and establish a baseline for management. Identifying the BCR::ABL1 transcript type is also important for subsequent quantitative molecular monitoring [5].

Tyrosine kinase inhibitors (TKIs) have transformed the treatment of chronic-phase CML, making long-term molecular surveillance and medication tolerability essential components of care [1,5]. We report a 30-year-old woman whose persistent leukocytosis, thrombocytosis, constitutional symptoms, and aquagenic pruritus led to a diagnosis of chronic-phase CML. Despite a favorable early molecular response to imatinib, clinically significant gastrointestinal intolerance required a change in therapy.

CASE PRESENTATION

A 30-year-old woman was referred to hematology in May–June 2026 after repeated complete blood counts revealed leukocytosis and elevated platelet counts. She reported longstanding generalized itching that worsened during or after showering, excessive sweating with drenching night sweats, and marked fatigue. An empiric course of antibiotics had been associated with no significant

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improvement in her white blood cell count.

The initial complete blood count in May 2026 showed a white blood cell count of 25.3 × 10^3/µL, hemoglobin 14.4 g/dL, mean corpuscular volume (MCV) 100 fL, and platelet count 453 × 10^3/µL. Repeat testing in June continued to show leukocytosis. On June 10, the white blood cell count was 30.54 × 10^3/µL, hemoglobin 12.2 g/dL, MCV 100.3 fL, and platelet count 404 × 10^3/µL. Differential counts showed neutrophilia, circulating myelocytes and metamyelocytes, and basophilia. A peripheral smear showed leukocytosis with a granulocytic left shift, thrombocytosis, and approximately 0-1 blast per 100 nucleated cells. The persistent leukocytosis, together with the presence of immature granulocytes and basophilia, raised concern for an underlying myeloproliferative neoplasm.

Bone marrow aspirate and biopsy obtained in June 2026 showed a markedly hypercellular marrow (approximately 95% cellularity) with granulocytic predominance and a granulocytic-to-erythroid ratio of approximately 7:1. Erythropoiesis was relatively decreased, whereas megakaryopoiesis was increased, with hypolobated and monolobated forms. Blasts were not increased: CD34 immunostaining identified approximately 1% blasts, and flow cytometry showed 1.1% blasts without a significant aberrant blast population. These morphologic findings supported a chronic-phase myeloproliferative neoplasm.

Quantitative reverse-transcription polymerase chain reaction (RT-PCR) on peripheral blood detected both major-breakpoint p210 BCR::ABL1 transcripts: e14a2 (b3a2) at 32.614% and e13a2 (b2a2) at 4.457%; the p190 e1a2 transcript was not detected. Conventional cytogenetic analysis was nondiagnostic because insufficient metaphases were available. The integrated morphologic and molecular findings established chronic-phase BCR::ABL1-positive CML. The treating hematologist recorded a Sokal score of 0.5, corresponding to low risk. Abdominal ultrasonography showed a spleen measuring 10.2 × 4.5 × 10.1 cm (reported volume approximately 243 cm^3) without reported splenomegaly. Serial BCR::ABL1 transcript measurements are summarized in Table 2.

Imatinib was initiated at 400 mg orally once daily in approximately July 2026. Initial adverse effects included diffuse joint pain, stiffness, and aches that affected daily activities, for which diclofenac was used as needed. Serial laboratory tests showed a substantial decline in leukocyte and platelet counts, accompanied by transient leukopenia and anemia. By July 29, the white blood cell count had fallen to 3.25 × 10^3/µL, hemoglobin to 10.8 g/dL, and platelet count to 178 × 10^3/µL. These findings were monitored without a dose reduction at that time. The patient subsequently reported reduced itching and sweating, and her joint symptoms gradually improved. Selected complete blood count results during evaluation and treatment are summarized in Table 1.

Molecular monitoring in August 2026 detected e14a2 at 1.5121% and e13a2 at 0.1196%, indicating a substantial decline from pretreatment levels and achievement of an early molecular response. Repeat testing in October 2026 showed e14a2 at 0.1009% and e13a2 reported as <0.0032%. The treating physician described a favorable molecular response; however, a sustained major molecular response had not yet been established by the available results.

Despite this response, the patient developed increasingly troublesome loose stools, at times occurring up to six times daily with mild cramping and occasional fecal incontinence. Symptoms affected food intake and daily functioning and persisted despite a trial of loperamide. Imatinib was temporarily withheld while stool testing for *Clostridioides difficile* was requested; the result was not available in the reviewed records. After a transient improvement and rechallenge, persistent gastrointestinal intolerance led to discontinuation of imatinib and prescription of asciminib 40 mg twice daily in late October 2026.

At the October 30, 2026 follow-up, the patient reported approximately eight days of asciminib use without new concerns. Following discontinuation of imatinib, her diarrhea had resolved, bowel movements occurred no more than twice daily, and she described improved energy. A subsequent virtual follow-up on November 4, 2026 documented continued asciminib therapy and resolution of loose stools. Her October 21 complete blood count showed a white blood cell count of 5.48 × 10^3/µL, hemoglobin 12.6 g/dL, and platelet count 232 × 10^3/µL. Ongoing hematology follow-up and molecular surveillance were planned; longer-term outcomes on asciminib were not yet available.

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Table 1. Selected Complete Blood Count Results During Evaluation and Treatment

Parameter	May 21	June 10	July 7	July 29	Aug 26	Oct 21
WBC (×10³/μL)	25.3	30.54	24.66	3.25	4.34	5.48
Hemoglobin (g/dL)	14.4	12.2	12.2	10.8	11.7	12.6
Platelets (×10³/μL)	453	404	481	178	165	232
MCV (fL)	100.0	100.3	104.6	105.9	104.9	102.9

Values represent selected recorded dates in 2026. WBC, white blood cell count; MCV, mean corpuscular volume.

Table 2. Serial BCR::ABL1 p210 Transcript Measurements

Specimen date	e14a2 (b3a2), %	e13a2 (b2a2), %
June 2026 (baseline)	32.614	4.457
August 2026	1.5121	0.1196
October 2026	0.1009	<0.0032

Transcript percentages are displayed exactly as reported by the laboratories. Molecular response thresholds should be interpreted using validated International Scale reporting and the entire clinical trajectory, rather than summing separate transcript values without laboratory confirmation.

DISCUSSION

This case highlights three interrelated aspects of contemporary CML care: diagnostic vigilance in younger adults, integrated morphologic and molecular testing, and the distinction between treatment efficacy and tolerability.

Although CML is diagnosed much more frequently later in life, age 30 falls within a recognized, albeit smaller, group of affected patients. SEER data indicate that individuals aged 20-34 years account for 6.7% of newly diagnosed CML cases [2]. In the randomized CML Study IV, younger patients had distinct presenting features; however, the study's 16-29-year and 30-44-year subgroups should not be conflated when discussing a patient aged 30 [3]. This patient's modest-to-moderate leukocytosis, lack of reported splenomegaly, and nonspecific fatigue could have been interpreted as reactive findings if considered in isolation. The combination of a persistent granulocytic left shift, basophilia, and thrombocytosis, together with pruritus and night sweats, justified evaluation for a myeloproliferative neoplasm. A structured approach to abnormal blood counts emphasizes repeat testing, peripheral smear examination, and hematology assessment when left-shifted leukocytosis remains unexplained [4]. The documented evaluation supports diagnostic vigilance; it does not establish that a diagnostic error or prolonged delay occurred.

The diagnostic workup also demonstrates the value of molecular confirmation when conventional chromosome analysis is inconclusive. The markedly hypercellular marrow, increased granulopoiesis, atypical megakaryocytes, and low blast percentage supported chronic-phase disease, while BCR::ABL1 testing identified the defining molecular abnormality. Current European

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LeukemiaNet recommendations emphasize transcript identification at diagnosis, baseline marrow and cytogenetic evaluation when feasible, and standardized quantitative PCR monitoring thereafter [5]. Poor culture growth prevented a fully informative conventional karyotype in this patient; it should not be described as a negative Philadelphia chromosome result. The detected e13a2 and e14a2 transcripts were therefore central to establishing and monitoring her diagnosis.

The course after imatinib initiation underscores the clinical difference between resistance and intolerance. Both reported p210 transcript measurements decreased substantially during treatment, and her blood counts normalized after an initial period of leukopenia and anemia. Molecular testing in August 2026 demonstrated a substantial reduction in both p210 BCR::ABL1 transcript levels, indicating a favorable early response to imatinib. However, because testing was performed before the standard three-month assessment, these findings should be interpreted as an early molecular response rather than a formal three-month milestone evaluation [5]. In contrast, gastrointestinal symptoms became progressively disruptive. Diarrhea, musculoskeletal symptoms, and fatigue are recognized adverse effects of imatinib and other TKIs [6]. Expert recommendations advise supportive care and careful monitoring for toxicities, while considering treatment interruption, dose adjustment, or an alternative TKI when adverse effects cannot be adequately controlled [7].

In a disease commonly requiring prolonged treatment, adverse effects should be assessed not only by laboratory severity but also by their effect on daily life. A quality-of-life study of patients receiving long-term imatinib found that younger adults and women could experience particularly substantial functional limitations [8].
Although the patient in this report was treated for only a few months before switching, her diarrhea, reduced oral intake, fatigue, and difficulty attending work illustrate why tolerability must be addressed even when molecular results are encouraging. The clinician's decision to change therapy was prompted by intolerance rather than documented molecular resistance.

Asciminib inhibits BCR::ABL1 through the ABL myristoyl pocket rather than the ATP-binding site. In ASC4FIRST, patients receiving asciminib for newly diagnosed chronic-phase CML achieved higher rates of major molecular response at 48 weeks than those receiving investigator-selected TKIs [9]. The US Food and Drug Administration expanded asciminib's indication to newly diagnosed Philadelphia chromosome-positive chronic-phase CML in October 2024 [10]. These data provide context for its selection, but they cannot demonstrate that it is superior for this particular patient. At the October 30, 2026 follow-up, the patient reported approximately eight days of asciminib treatment without new adverse effects. Her diarrhea had substantially improved, and she reported improved energy following imatinib discontinuation. However, this short follow-up period was insufficient to establish sustained asciminib tolerability or continued molecular response.

Several limitations should be acknowledged. Conventional cytogenetic assessment was incomplete because too few metaphases were available; the result of the ordered *Clostridioides difficile* test was not found in the reviewed material; and only short-term clinical follow-up after asciminib initiation was available. Moreover, the October e14a2 result of 0.1009% was marginally above the ≤0.1% International Scale cutoff for major molecular response, and the longitudinal interpretation of separately reported transcripts should be confirmed with the laboratory [5]. Continued complete blood count evaluation, serial BCR::ABL1 testing, and clinical assessment of treatment-related symptoms are therefore necessary. The principal lesson is not that CML at age 30 is unprecedented, but that persistent myeloid-pattern leukocytosis warrants appropriate investigation and that successful CML management must address both disease control and treatment burden.

CONCLUSION

This case highlights the importance of considering chronic myeloid leukemia in young adults with persistent, unexplained leukocytosis, particularly when accompanied by a granulocytic left shift, basophilia, thrombocytosis, or constitutional symptoms. Although CML is diagnosed more commonly in older adults, age alone should not delay peripheral smear evaluation, hematology referral, and appropriate BCR::ABL1 testing. In this patient, the diagnostic workup established chronic-phase CML, and imatinib produced a favorable early hematologic and molecular response. However, gastrointestinal intolerance substantially affected daily functioning and required a transition to asciminib. Early symptom improvement after the switch was encouraging, but long-term tolerability and sustained molecular control remain to be determined through ongoing follow-up. This case reinforces the importance of accurate diagnosis and individualized treatment decisions in CML management.

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DECLARATIONS

Ethical Approval: This case report describes the clinical presentation, diagnosis, and management of a single patient using information obtained during routine clinical care. No additional investigations or interventions were performed for research purposes.

Patient Consent for Publication: Written informed consent was obtained from the patient for the publication of this case report. Patient confidentiality has been maintained, and identifying information has been excluded from the manuscript.

Conflict of Interest: The authors declare no conflicts of interest.

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Data Availability: The relevant clinical findings and laboratory results are presented within the manuscript. Additional patient records are not publicly available to protect patient confidentiality.

Patient Perspective: A formal patient perspective was not obtained for this case report.

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